California Medicine
THE WESTERN JOURNAL OF MEDICINE

Studies on the Nature and Management of Psoriasis

EUGENE M. FARBER, M.D., *Stanford*

■ *Prevalence of psoriasis in Caucasians is estimated as 2 to 3 percent. Sound epidemiologic studies on a worldwide basis are needed to secure accurate prevalence rates for comparative purposes.*

Utilizing Stanford's psoriasis life histories records, the genetics of psoriasis has been explored by various means: statistical census data, pedigree analysis, and twin studies. This research suggests a multifactorial pattern of inheritance for psoriasis, implying that both genetic and environmental components are responsible for the manifestation of the disease.

At present it is not possible to point to any single causative factor. Some of the suggested areas for research include study of uninvolved skin, growth control in the psoriatic lesion, viral causes, immunological aspects, and lipid metabolism.

PSORIASIS IS a common skin disease affecting 2 to 3 percent of the Caucasian population.[1] Although there may be remissions, it must be regarded as incurable by any means now known. Unsightly and disfiguring, the lesions are a stigmatizing blight that causes emotional problems in many, and ruins the lives of persons with severe manifestations. Even persons whose lesions are not conspicuous are not secure, for without warning they may have severe exacerbations. It is an expensive disease, requiring lifelong treatment and care.[2]

The fundamental defect responsible for the development of the psoriatic lesion is still obscure, even though the literature is full of announcements of findings that are said to be specific. These supposedly definite features have been of various types, and at one time or another have pointed toward a multitude of etiological mechanisms. As time has passed, however, no specific attribute of the disease has been found.[3]

Clinicians have noted that psoriasis increases in severity following a number of phenomena. For example:

- Explosive exacerbations occur following

From the Department of Dermatology, Stanford University Medical Center.

This paper was presented as the Howard Fox Memorial Lecture at the New York Academy of Medicine, January 5, 1971.

Supported by grants from the John A. Hartford Foundation.

Submitted April 22, 1971.

Reprint requests to: Department of Dermatology, Stanford University Medical Center, 300 Pasteur Drive, Stanford, Ca. 94305 (Dr. E. Farber).

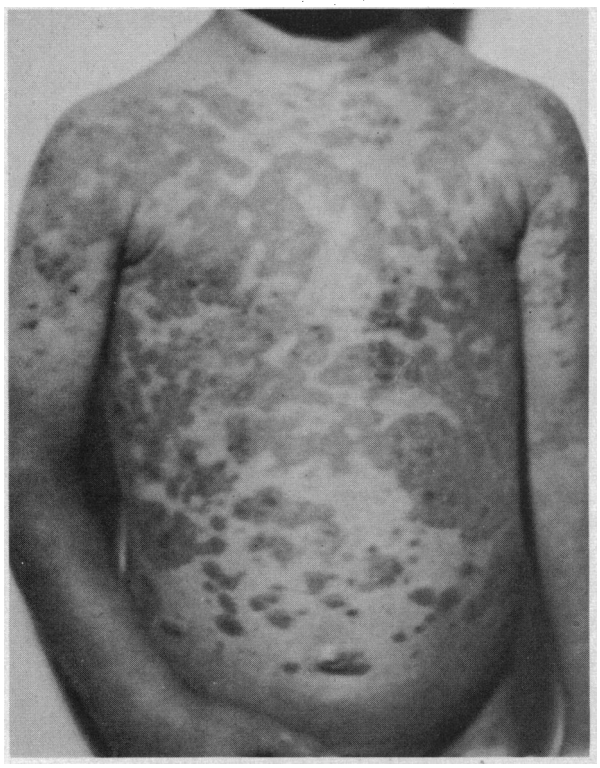

Figure 1.—A three-year-old male in whom psoriasis appeared after a streptococcal infection of the throat.

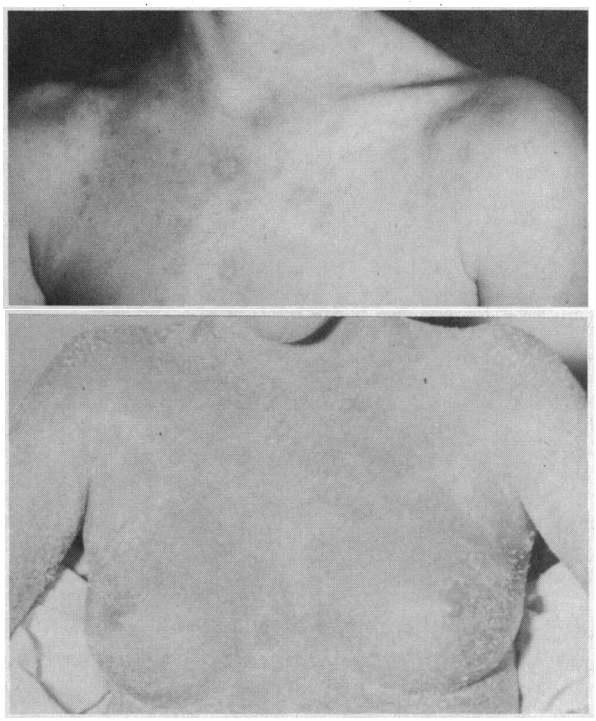

Figure 2.—Psoriasis began when the patient was eight years old and lesions were minimal (upper). Severe exacerbations occurred at the age of 18 years following a streptococcal infection (lower).

streptococcal infections in younger persons (Figure 1). Whyte and Baughman[4] reported that 17 out of 20 patients with acute guttate psoriasis had an antistreptolysin-O (ASO) titer greater than 200 Todd units.

• A person may have minimal psoriasis for ten years or more (Figure 2) and then unaccountably develop severe and widespread psoriasis. A careful history may reveal that the increase in severity of psoriasis, occurring later in the course of the disease, is due to injuries of various sorts to the skin.

• A 28-year-old woman experienced a severe flare of psoriasis after corticosteroid therapy was withdrawn (Figure 3), and a 40-year-old man, who had been receiving Declomycin®* for a secondary infection developed a phototoxic reaction following sun treatment for psoriasis (Figure 4).

• Rubber, dichromates and, sometimes, formaldehyde—all in the lining of shoes—may produce an inflammatory dermatitis on a contact-allergic basis which may trigger the development of lesions of psoriasis (Figure 5). Ointments

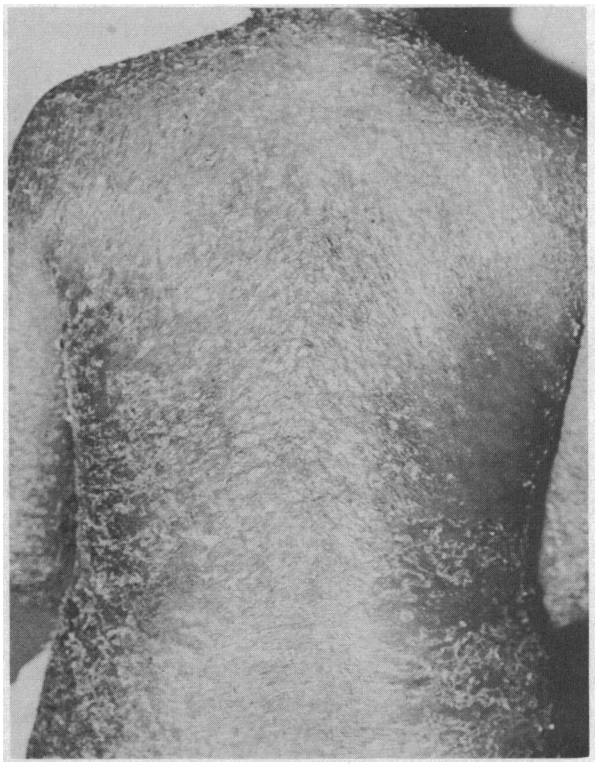

Figure 3.—A 28-year-old woman in whom severe exacerbations of psoriasis occurred after corticosteroid therapy.

*7-Chloro-6-demethyltetracycline; Declomycin® (Lederle).

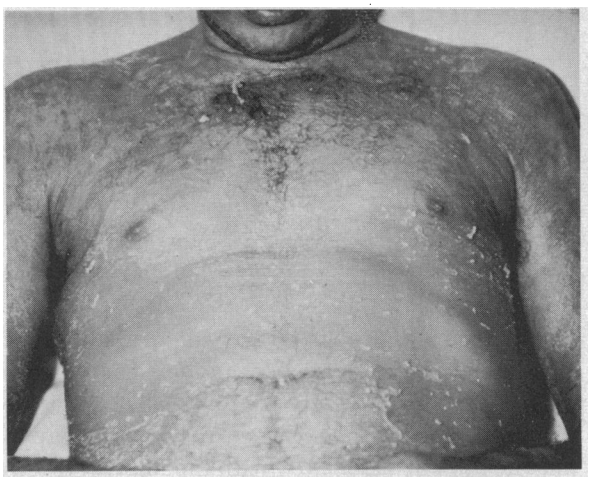

Figure 4.—A 40-year-old man, who had been receiving Declomycin® for a secondary infection, had a phototoxic reaction following sun treatment for psoriasis.

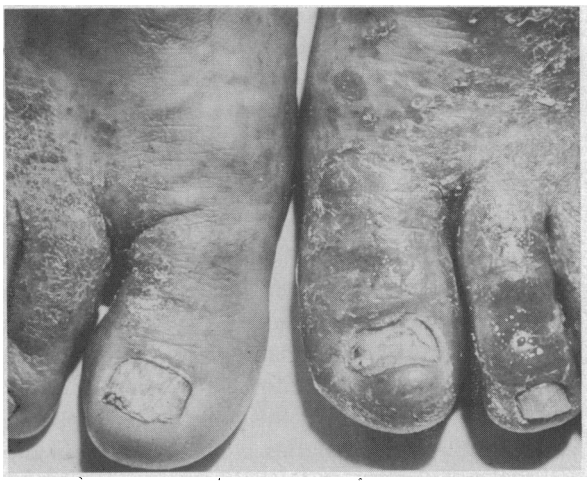

Figure 5.—In a 55-year-old man contact dermatitis from lining of shoes aggravated pre-existing psoriasis.

containing sensitizers also may produce isomorphic reactions and new lesions of psoriasis will appear.

Epidemiology and Genetics

Comprehensive epidemiological studies on the prevalence of psoriasis have been done in the past decade by Hellgren (1967)[5] and Lomholt (1963)[6] in the Scandinavian countries, and by Farber and his associates (1968)[7] in the United States.

Epidemiological studies reveal the frequency of psoriasis in a population and provide information to seek the causative agents for this disease. Reports in the literature on the prevalence of psoriasis in the general population have been based on the percentage of psoriatic patients found as in-patients or out-patients of hospitals or dermatology clinics.[1]

Prevalence

Generally, it has been observed that psoriasis occurs with relative frequency among Caucasians, less so in Mongoloids, and rarely in Negroes. In attempting to evaluate the differences in the frequency of psoriasis among different racial groups, Farber, Grauer and Zaruba[8] surveyed racial occurrence in groups throughout the world. They point out that in evaluating prevalence rates the influence of varying factors should be considered: genetics, climate, diet, degree of skin pigmentation, socio-economic background, and the like.

Various observers report the frequency of psoriasis in Caucasians as ranging from 0.1 to 2.84 percent;[1] Farber and Peterson[9] approximate the occurrence of psoriasis at 1 percent to 2 percent in the United States, and if all mild cases were included it might be as high as 3 percent. Psoriasis is more common among the Negroes than is ordinarily recognized. Kenney[10] studied 3,860 diagnoses of consecutive Negro private patients in this country from his dermatologic files. There were 27 (less than 1 percent) with psoriasis. Among 1,230 new patients, belonging to different tribes in Kenya, who had skin diseases 2.6 percent were diagnosed as having psoriasis.[11] It would appear that the occurrence of psoriasis is far higher in Africans than is usually reported. Lomholt[12] indicates that in the Dermatology Clinic of the Mulago Hospital of Uganda, psoriasis is quite common. Although no accurate figures are available, he sees one or two new psoriatic patients each time he visits the skin clinic. He said that the clinical feature is different from that which he observed in Scandinavia. (See Figure 6.)

In personal communications[13] from Indian Service Hospitals in Minnesota, Nebraska and North and South Dakota, ten cases of psoriasis in 38,400 Indians were reported. My only clinical experience with an American Indian with psoriasis was with a 53-year-old full-blooded Apache* man (Figure 7), who had widespread psoriasis.[14] Convit[15] examined 25,915 members of the general population in 95 small rural areas of Bolivia, Ecua-

*Referred by James Arnold, M.D., Medical Officer on the White-river Indian Reservation, Arizona (1968).

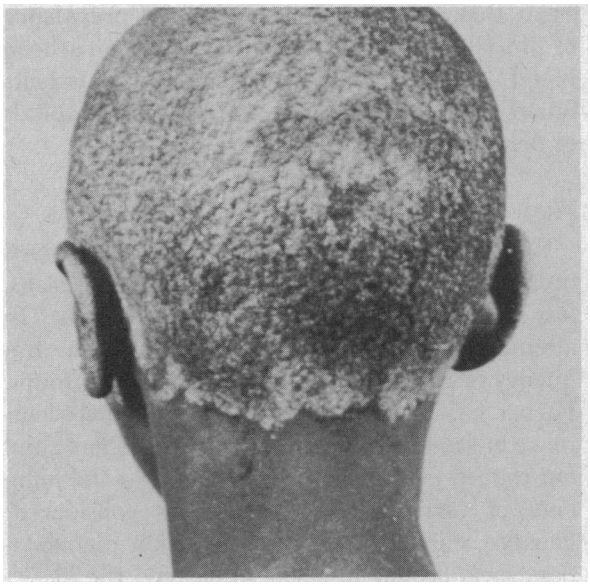

Figure 6.—An African Negro male from Uganda with psoriasis of the scalp. (Courtesy of Gunnar Lomholt, M.D.)

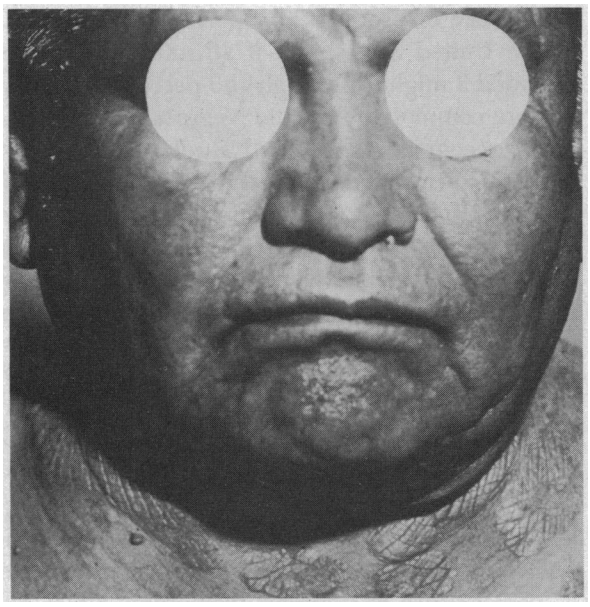

Figure 7.—A 53-year-old male Apache Indian with widespread psoriasis.

dor, Peru, and Venezuela. The locations varied in altitude from the torrid zones at sea level to temperate mountain areas at 10,000 feet. Not a single case of psoriasis was observed in the entire sample.

Psoriasis is said to be less frequent in the Japanese than in Caucasians. Watanabe et al[16] said that before World War II psoriasis accounted for 0.36 percent of all skin diseases in Japan but the incidence had risen to 0.64 percent in 1958. Precise epidemiological data are not available, but Japanese investigators report an increasing prevalence of psoriasis in their country.[17] The increase in the number of persons seeking care in Japan may be directly associated with the improved economic conditions in that country and the greater number of dermatologists in practice.

TABLE 1.—*Percentage of Relatives of Psoriatics who have Psoriasis*

Investigator	*Psoriasis in families (Percent)*	*Psoriatics in sample (Number)*
Hoede 1931[19]	39	1,437
Hellgren 1967[5]	36	450
Farber et al 1968[7]	36	2,144
Lomholt 1963[6]	91[a]	312

[a]This is an inbred population.

No cases of psoriasis have been found in native inhabitants of Fiji.[8]

Farber[18] personally examined more than 500 patients in American Samoa and did not find psoriasis in any of them, nor has any physician in practice there reported a Samoan with this disease.

Genetics

In the Stanford Psoriasis Life Histories Research Unit, approximately 7,000 records of persons with psoriasis are on file. From this file data has been gathered on the occurrence of psoriasis in the families of psoriatic patients. It has long been known that psoriasis "runs in families." Many studies have shown that the prevalence of psoriasis in families of psoriatics is greater than in the general population.

In about one-third of the instances, first, second, and third degree relatives of psoriatics have psoriasis (Table 1).

Familial concentration of a disease raises the question of genetic factors contributing to its manifestation. One can investigate the genetics of a trait by various means including statistical census studies, pedigree analysis, and twin studies.[20]

It is now recognized that many diseases not inherited in a simple manner have some hereditary basis. A genetic component has long been suspected for such conditions as diabetes, rheumatoid arthritis and psoriasis, but genetic analyses do not support inheritance by single gene differences.

Although many investigators agree that the high occurrence of psoriasis in families leads to the assumption that genetic factors play an etiologic

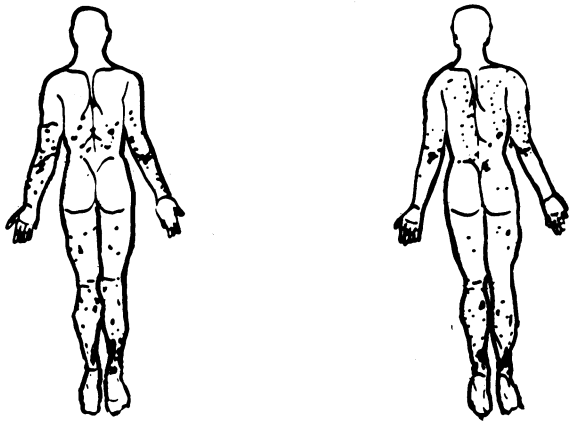

Figure 8.—Schematic illustration of geographic distribution of psoriasis in 18-year-old monozygotic male twins, showing similarity of pattern.

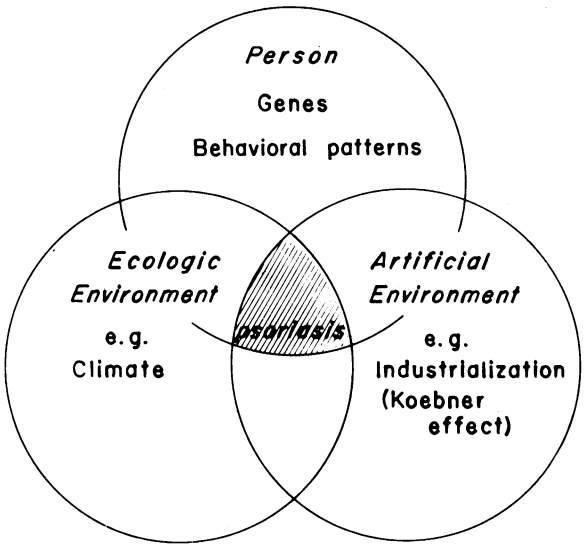

Chart 1.—A Venn[23] diagram illustrating the interaction of genetic and environmental components. The intersection of the three circles represents the presence of psoriasis. The top circle represents the person himself with a genetic and behavioral predisposition for psoriasis and the other two circles represent factors in the natural and artificial environments that precipitate the psoriatic lesion.

role, there is no agreement on the mode of inheritance. The interpretation of the observations ranges from simple dominance to digene recessivity.[21]

The prevailing theories of the modes of inheritance of psoriasis are:

- simple autosomal dominant with incomplete penetrance;
- double autosomal recessive;
- multifactorial.

Farber and Nall[22] in reviewing data on 40 pairs of monozygotic twins, noted that 27 pairs were concordant for and 13 pairs were discordant for psoriasis. Of 39 pairs of dizygotic twins, nine were concordant and 30 discordant. That not all monozygotic twins were concordant would indicate that other factors—environment, for example—are also influential. Why 13 of the monozygotic twin pairs were discordant for psoriasis requires careful study and analysis.

There are several pairs of both monozygotic and dizygotic twins in our different population samples. Two male monozygotic twins developed psoriasis at the same time with similar patterns of coverage over the surface of the body area (Figure 8). Zygosity diagnosis revealed they had identical blood types.

In a study of 698 probands with psoriasis, Watson et al[20] found from a mating analysis that:

a) there were 1,089 siblings from families in which neither parent had psoriasis; 7.8 percent of these siblings had psoriasis;

b) 16.4 percent of the siblings (256) had psoriasis when *one* parent was affected;

c) 50 percent of the siblings (12) had psoriasis when *both* parents had psoriasis. (It should be noted that the last figure is too small a sample to be statistically significant.)

In autosomal dominant inheritance, 50 percent of the siblings from one affected parent would be expected to have psoriasis. In autosomal recessive inheritance, one-quarter of the siblings of probands would be expected to be affected if neither parent is affected, and all of the siblings when both parents are affected.

Our figures are much lower than those expected from simple autosomal dominance and recessivity or sex-linked inheritance, and the mode of inheritance cannot be explained by any of these.

Our family studies would suggest a multifactorial pattern of inheritance for psoriasis. This implies that multiple genes and environment are responsible for the clinical appearance of the disease. The concept of genetic and environmental components interacting in the manifestation of psoriasis is illustrated in Chart 1.

Suggested Fields for Psoriasis Research

Psoriasis, like many another disease, has remained an enigma. The cause is unknown. The factors which are known are: the skin is the only tissue involved; there is a familial-genetic background; lesions are symmetrical; nails are in-

volved; there is no diagnostic test; there are no metabolic abnormalities; and injury to the skin can precipitate a psoriatic lesion.

Psoriasis is characterized by thick lesions covered with scales; there is much mitotic activity in the epidermis as well as accelerated chemical processes. Parakeratosis, vascular changes, and inflammation are thought to be secondary phenomena. Rapid epidermal cell growth is due to rapid cell turnover, but this is not qualitatively different from other types of rapid epidermal growth.

At present it is not possible to point to any single causative factor. Therefore, it is essential to review every possibility for this particular growth disturbance. Some fields of investigation are more promising than others:

Study of Uninvolved Skin

Study of the predisposed, but uninvolved, skin in psoriasis (particularly in the early stages of evolving changes) is more likely to yield information concerning the nature of the psoriatic process than could be expected from further study of psoriatic plaques themselves. The cause should be sought in a deviation of the growth-control mechanism rather than in the rapidly growing epidermis *per se*.[24]

If it is possible to identify the presence of some predisposing feature of the symptom-free skin in patients destined to have psoriasis, it will open the door to significant research.

There is no animal model for psoriasis. An appropriate human model for the study of psoriasis is the patient himself. The symptomless skin of a psoriatic patient can be used for research by inducing lesions through experimental injury.

Growth Control in the Psoriatic Lesion

Psoriasis is a disturbance characterized by the rapid growth of epithelial tissue. All palliative therapy is designed to keep epidermal growth at its normal level, whereas curative therapy is designed to restore normal growth control. Not known is how the growth control is operative or where it is located. Therefore, research cannot be narrowed down too much, because many areas remain to be explored.

When normal skin is stripped with Scotch tape, its epidermis grows faster, indicating that a regulating factor has been removed.[25] What keeps normal epidermis in check? In psoriatic patients, injury to symptomless skin triggers epidermal growth that continues longer than that of normal epidermis following injury. This isomorphic response can be used as a model to study the evolution of the psoriatic lesion, by histologic means or measurements of the variety of biochemical changes in the evolving lesion.

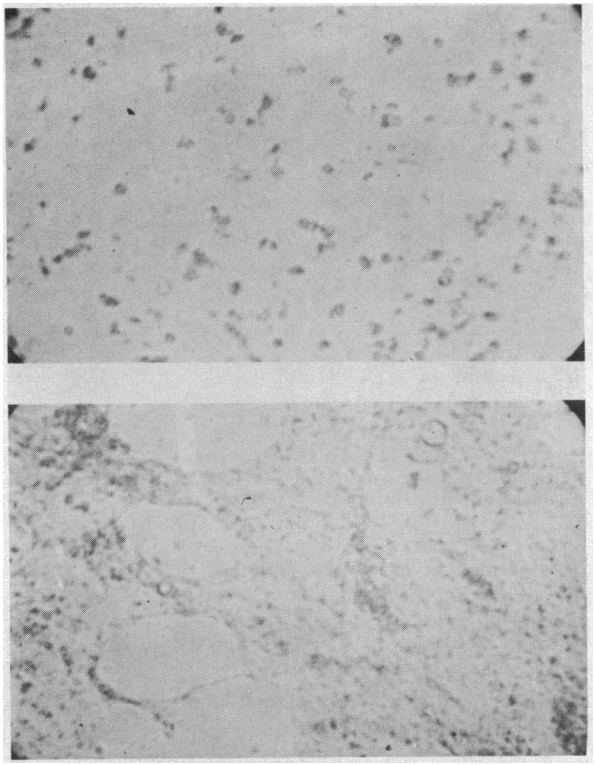

Figure 9.—Epidermal cell growth. Top frame shows control in which isolated epithelial cells were grown in standard media for five days. Lower frame shows epithelial cells grown in conditioned media from rabbit dermal fibroblasts for five days.

Useful information may be provided by detailed studies of the nucleus in symptomless skin, using quantitative histochemistry or autoradiography for early changes in deoxyribonucleic acid (DNA) synthesis in evolving lesions.[26]

Feedback inhibition of DNA synthesis by end-products of nucleic acid metabolism is a possibility that needs exploration. The induction and repression of enzymes of nucleotide synthesis in the basal cell population, and the allosteric control of DNA and ribonucleic acid (RNA) synthesis are approaches that properly could be applied to the skin epithelial cell.

Karasek[26] has studied the role which the dermis plays in the regulation of epithelial cell growth. The top photograph of Figure 9 shows a control

in which isolated rabbit epithelial cells were grown in the presence of the standard media for five days. In the lower photograph are epithelial cells grown in the presence of conditioned media from rabbit dermal fibroblasts for five days; the initial growth of the control slide has stopped. While in the presence of conditioned media, growth was definitely stimulated.

To clarify this phenomenon in relation to psoriasis, study of the isolation and characterization of the cues produced or stored by the dermis should be undertaken, and an analysis of the way in which these cues interact with the epithelial cell to increase or repress mitosis should be made.

It can logically be argued that epithelial growth response in psoriasis is a result of dermal factors. This is a field for useful future research in psoriasis.

Viral Research

There is evidence that viruses cause disturbances in cell growth, and that they modify or control cell growth.[27] If there is a virus in psoriasis, it has not been identified. A case can be made for the possibility of a viral factor in psoriasis. One could postulate a viral DNA as well as a tissue DNA existing in the cell nucleus, with the former modified by successful treatment, the mammalian DNA continuing to function with resumption of normal cell growth.

It is well known that heat or light activates herpes simplex virus. Is this a Koebner reaction? In psoriasis, injury or environmental changes may result in a Koebner reaction. Is the isomorphic response in psoriasis a result of a virus-provoking cause?

Immunologic Aspects

There is no clear-cut evidence to support an immunologic basis for psoriasis, though several reports in the literature have pointed to a possible immunologic disturbance.[4,6,28]

In the Stanford Psoriasis Questionnaire Survey of 2,144 psoriatic persons,[7] 14 percent indicated that they had had either asthma or hay fever—about the same prevalence as for atopy in nonpsoriatic persons.

Immediate hypersensitivity in patients with psoriasis is no different from that in controls, and there is no hard evidence for differences in delayed hypersensitivity.

In support of immunologic factors are some clinical observations such as the appearance of guttate lesions after streptococcal infection, and remission following measles.[4]

There have been several reports of measles altering the course of psoriasis. Lomholt[6] investigated the effect of measles on psoriasis and found cases of severe guttate eruptions of psoriasis following measles. He also found, however, complete disappearance of psoriatic lesions in seven patients during and following measles. This led to speculation by some observers that immunologic factors might play a role in the pathogenesis of psoriasis, since the lymphoid system is somewhat depressed during infection with rubeola.

There is insufficient information about possible defects in the cellular immune response or the mediators of inflammation. Such studies should be carried forward with the most modern and sophisticated of methods.

Lipids

Is there a case for faulty lipid synthesis? At present, changes in membrane structure are thought to be responsible for the abnormal behavior of many types of cells.[29] (A defect in the cell membrane structure includes the outer membrane as well as the internal membranes of the endoplasmic reticulum.)

These membrane changes can be induced by viruses or by a variety of chemicals. Rubin's concept[30] of carcinoma cells is related to the synthesis of abnormal protein in cells that changes the cell membrane and cell growth.

Whether or not there is a defective membrane synthesis in psoriasis deserves a careful look.

Treatment

Psoriasis requires treatment because of pruritis, disfigurement and frequently pain. Treatment is very often symptomatic. More recently, fundamental science has been reflected in varying forms of experimental therapy. It is conceivable that with much research effort, a specific agent could be found which, when applied to the lesions, would control psoriasis much as insulin controls diabetes.[29]

An effort should be made to find antimetabolites which will have a suppressive effect on psoriasis without the hazards of toxicity associated with systemic administration. Since psoriasis and can-

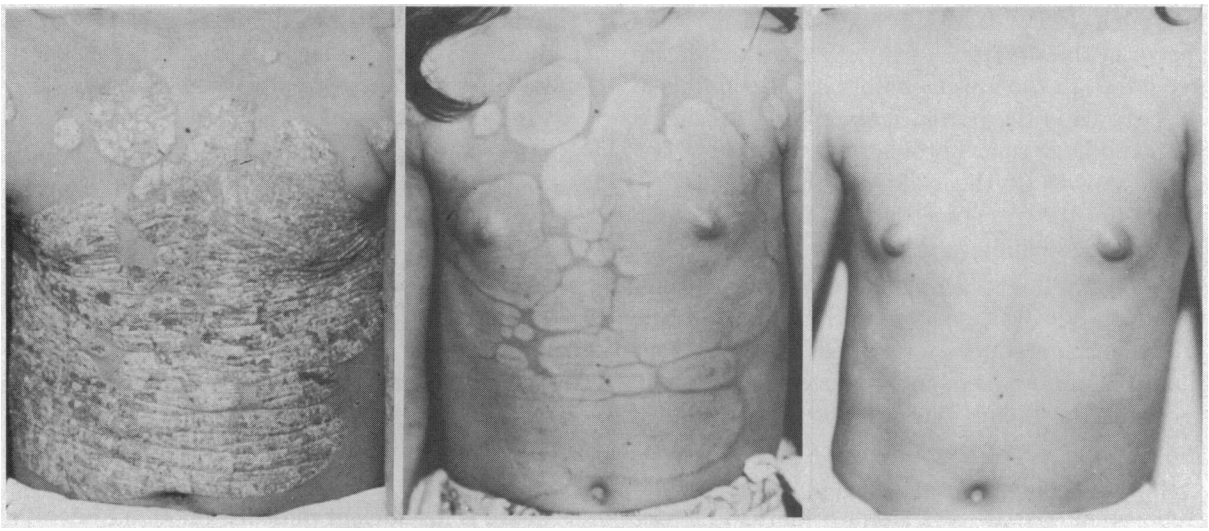

Figure 10.—Psoriatic lesions before treatment with anthralin (left) one week later (center) and three months later.

cer have the common characteristic of cellular hyperplasia and increased metabolic and mitotic activity, antimetabolites successful in controlling cancer should be tried in psoriasis.

When a distinct suppression of the psoriatic process is noted from the application of a test substance, a detailed analysis of the mechanism involved should be performed. These could include electronmicroscopy, tritiated thymidine uptake, p-32 uptake, and mitotic counts, before and after therapy.

The finding of a practical, non-toxic and effective topical treatment would be of immeasurable benefit to the estimated 4 to 6 million patients in the United States who have this chronic skin disease.

Available now are only a few drugs—all palliative, and all involved with side reactions:

Crude coal tar, Anthralin, corticosteroids, retinoic acid
5-fluorouracil, 6-mercaptopurine, 6-diazo-5-oxo-L-norleucine, hydroxyurea
Actinomycin D, Puromycin, Podophyllin

There is hope, through proper studies, of improving the situation. Anthralin, for example, is a drug that has been long known but not sufficiently appreciated.[32,33] The relapse rate, however, is nearly 100 percent; and the treatment is messy and irritating (Figure 10). Nonetheless, it is extremely effective when applied carefully and under supervision.

Crude coal tar has been widely used in the treatment of psoriasis for more than 50 years. In these before-and-after photographs (Figure 11) a striking remission is seen; but almost all patients relapse, and not all do as well as the patient shown.

Another patient was treated on her left side with an aqueous solution of 0.05 percent nitrogen mustard,[34] applied daily for ten days to a small area of psoriasis on her back (Figure 12). The treated area completely cleared. However, 90 percent of patients treated with nitrogen mustard have severe reactions of delayed hypersensitivity if treatment is continued. The drug is also potentially toxic and, therefore, not suited to general use. A search for a non-toxic drug for psoriasis should continue with undiminished energy.

Conclusion

As new advances are made in the basic biology of cell growth, there will be a greater understanding of ways of controlling the rapid epidermal cell growth that is characteristic of psoriasis.

To solve the problem of psoriasis, it is probably necessary to:

- Develop interdisciplinary studies involving dermatologists, skin biologists, pharmacologists, and other basic scientists.
- Establish psoriasis centers that could participate in research with other medical institutions as well as with each other. Centers for psoriasis could act as a resource for a region of the country.

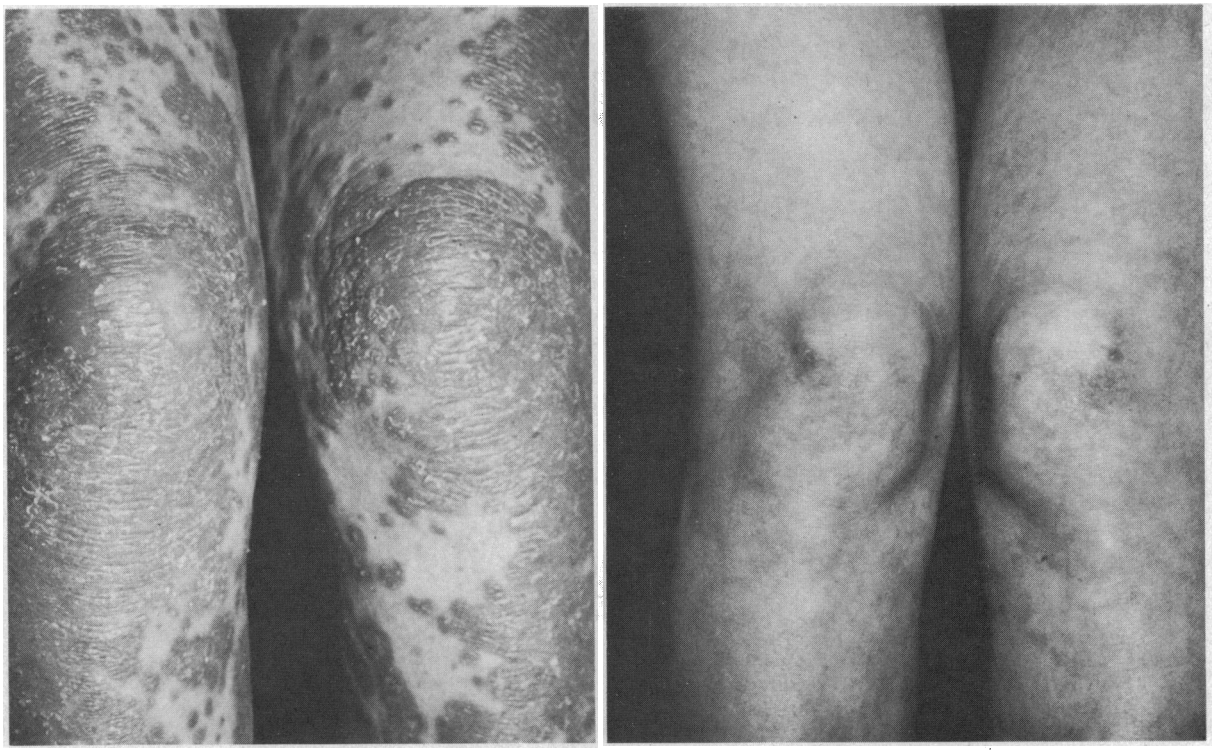

Figure 11.—Psoriatic lesions before treatment with 2 percent crude coal tar in an ointment base (left) and six weeks after treatment.

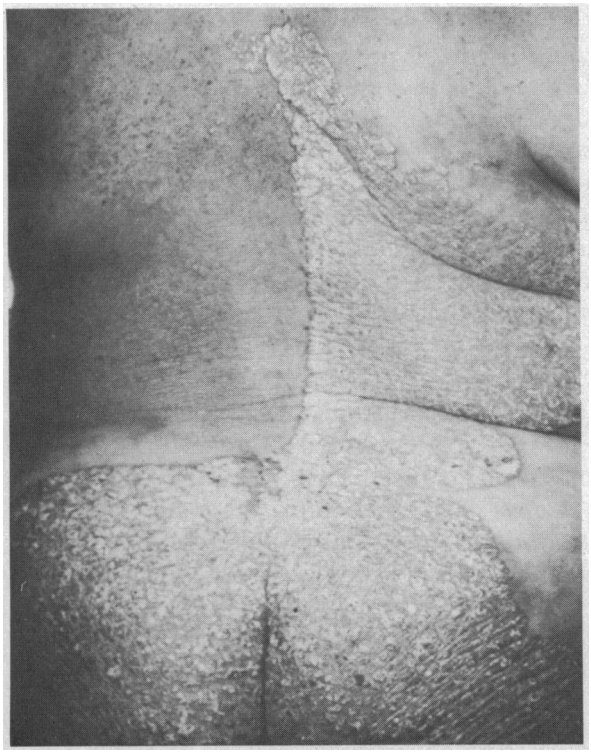

Figure 12.—Psoriasis treated with nitrogen mustard. This photograph taken three months after a ten-day course of treatment shows clearing in the treated area.

Their expertise in the clinical care, research, and education should link them with all dermatologists in their area. Such centers can provide information for all dermatologists who wish to visit and learn. Cooperating psoriasis centers and dermatologists can share in on-going research and have available the reports of their findings before publication.

The resources in support of psoriasis research today are insufficient. More investigators and funds are needed, for without both, research ideas cannot be implemented.

ACKNOWLEDGEMENT: The author expresses his appreciation to Alvin Cox, M.D., and to Marvin Karasek, Ph.D., for their valued suggestions.

REFERENCES

1. Baker H: Epidemiological aspects of psoriasis and arthritis. Brit J Derm 78:249-261, 1966

2. Farber EM, Roth RJ: Course and care of psoriasis. Mod Med Feb:100-106, 1965

3. Farber EM, Cox AJ: Biology of psoriasis. J Invest Derm 49:348-357, 1967

4. Whyte HG, Baughman RD: Acute guttate psoriasis and streptococcal infection. Arch Derm 89:350-356, 1964

5. Hellgren L: Psoriasis—The Prevalence in Sex, Age and Occupational Groups in Total Populations in Sweden—Morphology, Inheritance and Association with Other Skin and Rheumatic Diseases. Stockholm, Almquist & Wiksells, 1967

6. Lomholt G: Psoriasis—Prevalence, Spontaneous Course and Genetics—A Census Study on the Prevalence of Skin Diseases on the Faroe Islands. Copenhagen, BEC Gad, 1963

7. Farber EM, Bright RD, Nall ML: Psoriasis—A questionnaire survey of 2144 patients. Arch Derm 98:248-259, 1968

8. Farber EM, Grauer F, Zaruba F: Racial incidence of psoriasis. Cesk Derm 40:289-297, 1965

9. Farber EM, Peterson JB: Variations in the natural history of psoriasis. Calif Med 95:6-11, 1961

10. Kenney JA: Management of dermatoses peculiar to Negroes. Arch Derm 91:126-129, 1965

11. Verhagen ARHB, Koten JW: Psoriasis in Kenya. Arch Derm 96:39-41, 1967

12. Lomholt G: Personal communication

13. Farber EM: Unpublished data

14. Farber EM: Unpublished data

15. Convit J: Investigation of the incidence of psoriasis among Latin American Indians. Proc 12th Internat Congr Derm 2:196-199, 1963

16. Watanabe S, Ninomiya S, Minagawa S, et al: Acta Derm (Kyoto) 54:146, 1959

17. Yasuda T, et al: Personal communications

18. Farber EM: Unpublished data

19. Hoede K: Unwelt und Erblichkeit bei der Entstehung der Schuppenflechte. Wursb Abh Med 27:211-254, 1931

20. Watson W, Cann HM, Farber EM, et al: The genetics of psoriasis. Presented at American Medical Association annual meeting, Chicago, Ill., June 22, 1970. (To be published)

21. Aschner B, Curth HO, Gross P: Genetic aspects of psoriasis. Acta Genet 7:197-204, 1957

22. Farber EM, Nall ML: Psoriasis in twins. (To be published)

23. Johnson KG: Epidemiology, *In* Kilbourne ED, Smillie WG. (Ed): Human Ecology and Public Health. Toronto, The Macmillan Co, 1969, pp 125-142

24. Cox AJ: The dermal-epidermal junction in psoriasis. J Invest Derm 53:428-435, 1969

25. Pincus H: Examination of the epidermis by the strip method—II. Biometric data on regeneration of the human epidermis. J Invest Derm 19:431-446, 1952

26. Karasek M: Personal communication

27. von Wettstein D, Lagerholm B, Zech H: Cellular changes in the psoriatic epidermis. Acta Dermato-Venereal 41:115-134, 1961

28. Landau JW, Gross BG, Newcomer VD, et al: Immunological response of patients with psoriasis. Arch Derm 91:607-610, 1965

29. Wilkinson DI, Farber EM: Free and esterified sterols in surface lipids from uninvolved skin in sporiasis. J Invest Derm 48:249, 1967

30. Rubin H: Overgrowth simulating factor released from Rous sarcoma cells. Science 167:1271-1272, Feb 27, 1970

31. Farber EM, Ferrington R: Treatment of psoriasis. Presented at Psoriasis Symposium, Toho University School of Medicine, Tokyo, Japan, Oct 19, 1970. (To be published)

32. Farber EM, Harris DR: Hospital treatment of psoriasis: A modified anthralin program. Arch Derm 101:381-389, 1970

33. Comaish S: Ingram method of treating psoriasis. Arch Derm 92:56-68, 1965

34. Zackheim HS, Farber EM, Cox AJ, et al: Effect of topical nitrogen mustard on psoriasis. (To be published)

ON WITHDRAWING STEROIDS

In patients with chronic ulcerative colitis who have been on steroids for more than five years, how soon after surgery do you stop the medication?

"I would suggest first that if you have to treat patients with steroids for long periods of time, you should use the medication every second day and allow a day for the adrenals to escape at least partially. In that way there is less adrenal suppression. In other words, if you plan to use 15 mg of prednisone a day, use 30 mg every second day instead. I see no difference in the control of the patient.

"Patients who take steroids for a prolonged period of time must be taken off very slowly, indeed. Perhaps these patients should have an ACTH stimulation before they are taken off and perhaps they should even have adrenal function studies, too. I think that you would be talking about a period of weeks. I would tend personally to taper patients off very slowly and watch them very carefully over a period of many weeks. If they were taking 20 mg of prednisone a day, I would cut the dosage down by no more than one tablet each month. If there were any suggestion of difficulty, I would stop and do adrenal function studies and be certain that the adrenals were able to respond."

—F. WARREN NUGENT, M.D., Boston

Extracted from *Audio-Digest Surgery*, Vol. 16, No. 15, in the Audio-Digest Foundation's subscription series of tape-recorded programs. For subscription information: 619 S. Westlake Ave., Los Angeles, Ca. 90057.